A model of the effects of cognitive load on the subjective estimation and production of time intervals

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Abstract

How and where the brain calculates elapsing time is not known, and one or more internal pacemakers or others timekeeping systems have been suggested. Experiments have shown that the accuracy in estimating or producing time intervals depends on many factors and, in particular, both on the length of the intervals to be estimated and on the additional, and unrelated, cognitive load required during the task. The psychological ‘attentional approach’ is able to explain the experimental data in terms of perturbations of a cognitive timer. However, the basic biophysical mechanisms that could be involved at the single neuron level are still not clear. Here we propose a computational model suggesting how the process to focus the attention on a non-temporal task could alter the perception of time intervals as observed in the experiments. The model suggests that an attention-based excitatory and/or inhibitory background synaptic noise, impinging on the pacemaker circuit, could represent both qualitative and quantitative features of the cognitive load. These effects are predicted to be independent of the number, location or specific implementations of the internal timing systems. © 2000 Elsevier Science Ireland Ltd. All rights reserved.

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1. Introduction

An accurate perception/calculation of time intervals is important in a number of everyday activities. Detecting deviations that could underlie malfunctioning or unexpected problems, the planning of actions between two events, or waiting for an event’s end, require the subjective calculation of time durations. Internal as well as external factors, such as personality traits (Orme, 1969), age (Wallach and Green, 1961), body temperature (Hancock, 1993), drugs use (Frankenhaeuser, 1959), and degenerative brain diseases (Nichelli et al., 1993), among many others, may affect it. Furthermore, experimental findings (Zakay, 1993; Zakay and Shub, 1998; Marmaras et al., 1995) suggest that the subjective calculation of a time interval depends on the length to be estimated or produced, and on the additional, and unrelated, cognitive load that diverts the attention from the elapsing time. Although the basal ganglia, the
cerebellum and selected cortical structures could be involved in the representation of temporal information (for reviews see Ivry, 1996; Gibbon et al., 1997), how and where the brain computes time durations is still essentially not known. From a psychological point of view, the attentional approach (Frankenhaeuser, 1959) has been shown to be consistent with the experimental findings. It postulates the existence of a cognitive timer that counts subjective time units. Because this timer demands attentional resources, time estimation should be a direct function of the amount of attention devoted to it. A neuronal circuit, including a pacemaker and additional control circuitry, has been suggested as cognitive timer (Treisman, 1963). Time units would be marked by the action potentials (APs) generated by the pacemaker, and the subjective time durations depends on the modulations in the pacemaker’s frequency. Although the attentional approach is able to explain the experimental data, the basic biophysical mechanisms that could be involved with these modulations are still not clear, and their investigation is the purpose of this work. The signal pathway, within the cognitive timer, most likely encompasses several neurons, involving either many cycles through circuits of a few cells or propagation through many serially connected neurons. Thus, we argued that the latency of the pacemaker APs and the factors affecting it might be among the mechanisms that alter, at the single neuron level, the calculation of subjective elapsed time. Here we propose a computational model suggesting how and to what extent these mechanisms could be responsible for the observed effects.

2. Methods

In order to keep the model and the interpretations of its results as simple as possible, only experiments based on prospective (as opposed to retrospective) paradigms have been taken into account. In prospective experiments, subjects know in advance that a time interval estimation/production will be required. Thus, the task can be carried out with minimum (if at all) interplay with additional activities such as memory searches or replay of memorized events that, on the other hand, are needed using retrospective paradigms.

The typical experimental findings that we have chosen as starting point of our model are shown in Fig. 1. To test the accuracy of time interval production as a function of interval length and concurrent cognitive load, Marmaras et al. (1995) asked subjects to produce time intervals of various lengths while doing tasks of increasing cognitive load (Fig. 1A). The intervals produced were systematically longer than controls (15, 30 and 60 s). Zakay (1993) earlier reported analogous findings for the production of a 12 s interval (Fig. 1B, closed symbols). However, shorter intervals resulted when subjects were asked to estimate the same interval (Fig. 1B, open symbols). It should be stressed that production and estimation of a time interval are two totally different tasks. To study time estimation, the experimenter himself starts and stops the experiment. When an interval production is requested, the subject himself/herself controls the start and stop signals. These kinds of experimental findings are currently interpreted by the psychological theories in terms of attention-induced perturbations of the pacemaker circuitry. In order to study what could be the biophysical mechanisms involved with these perturbations we modeled a simple pacemaker circuit

![Fig. 1. Typical experimental findings. Task complexity increases from left to right. (A) Time intervals produced under concurrent cognitive loads. Intervals of different lengths (15, 30 and 60 s) were tested. Data taken and redrawn from Marmaras et al. (1995). Task C0 is control condition. (B) Production or estimation of a 12 sec interval under cognitive loads of increasing complexity. Data taken and redrawn from Zakay (1993). Task ET is control condition.](image-url)
Fig. 2. (A) Pacemaker model. A suprathreshold (9 nS) synapse, on a proximal apical trunk (50 μm from the soma), was activated by either a somatic AP after a conduction delay of 45 ms or a 100-ms silent period. (B) Weak noise. Superposition of typical somatic membrane potential (the pacemaker output) from three simulations using different background noise conditions. The arrow indicates the time of synaptic stimulation in all cases. (C) Strong noise. Typical somatic membrane potential is shown for simulations using different background noise conditions.

(Fig. 2A). We assumed that a more or less complicated circuit of neurons, rather than a single neuron with intrinsic oscillatory properties, composes a pacemaker. There is no direct experimental evidence for the network connectivity or for the passive and active properties of the neurons involved with time processing. We have chosen to start from a realistic model of a pyramidal neuron, an ubiquitous kind of cell in the brain, modeled using 3D morphological data and accurate kinetics and distributions for the ionic channels (Migliore et al., 1999). We then added a simple feedback circuit. The latter was implemented as a suprathreshold (9 nS) synaptic contact, inserted on a proximal apical trunk (≥50 μm from the soma) and activated by a somatic AP after a conduction delay of 45 ms. Because the synapse elicited a somatic AP with a 5 ms latency, once started under control conditions the pacemaker generated a regularly spaced train of APs at 20 Hz. This value is consistent with indirect experimental estimates of the possible characteristic frequencies of an internal clock (Treisman et al., 1990). Finally, to transform the circuit into a pacemaker, the synapse was forced to be activated after a 100ms silent period, to model possible effects of network synchronization at the Theta rhythm. According to the current psychological models using a pacemaker, to estimate the interval between a start and a stop signal, an internal clock circuit just multiplies the number of output pulses from the pacemaker by an internal reference frequency, 20 Hz in our simulations. The model’s estimation of an nsec interval, was thus calculated from the number of pulses generated by the pacemaker during an nsec long simulation. To produce an interval, a simulation lasted until the pacemaker generated the expected number of APs. An nsec interval thus required to wait for n · 20 APs. The total simulation length was the model production of an nsec interval. Unless otherwise noted, average values of the produced/estimated intervals from ten simulations were used. In most cases, error bars (S.E.M.) were
smaller than symbols size or line widths and are not shown.

2.1. Computational details

All the simulations were carried out using the NEURON simulation program (version 4.2.1, Hines and Carnevale, 1997) on a DEC Alphaserver 4100 5/400 workstation. The details of the ionic channel kinetics and distribution, as well as the passive parameters of the model neuron, were presented elsewhere (Migliore et al., 1999). In our simulations we anticipated the use of many thousands of synaptic activation (several of which overlapping in time). Thus, the use of a detailed model for a synapse would result in prohibitively long simulation times. On the other hand, in this work there were no specific needs for a detailed synaptic model. For these reasons, we implemented a synaptic conductance using a simple kinetic scheme that resulted in the following set of ordinary differential equations:

\[
\frac{dK}{dt} = \alpha I - \beta K
\]

\[
\frac{dC}{dt} = \beta K - \epsilon C
\]

where a presynaptic current of \( I \) nA generates, through an intermediate reactant \( K \), a postsynaptic conductance of \( C \) \( \mu \)S. The following values for the rate constants were used: \( \alpha = 1.25 \frac{\text{mV}^{-1}}{\text{ms}^{-1}} \), \( \beta = 0.33 \frac{\text{ms}^{-1}}{\text{ms}^{-1}} \), \( \epsilon = 1 \frac{\text{ms}^{-1}}{\text{ms}^{-1}} \). The synaptic current (in nA) was then calculated as \( I_{\text{syn}}(t) = C(t)(v(t) - e_{\text{syn}}) \), where \( v(t) \) was the membrane potential (in mV) and \( e_{\text{syn}} \) the synaptic reversal potential (\( e_{\text{syn}} = 0 \) and \( -80 \) mV for excitatory and inhibitory synapses, respectively). The resting membrane potential of the model neuron was fixed at \( -65 \) mV. With these parameters, a presynaptic short (0.2 ms) \( I \) pulse of 0.02 nA resulted in a peak synaptic conductance of \( C \geq 1 \) nS. In the rest of the paper we will always report the peak synaptic conductance, rather than the strength of the presynaptic current pulse that generated it. Synaptic background noise was modeled with a random (poissonian) activation of an excitatory and/or inhibitory synapse at an average frequency of 200 Hz, with gaussian distributed peak conductances, in the proximal (50 \( \mu \)m from the soma) apical trunk. The NEURON model and simulation files are available from one of the authors (MM).

3. Results

Several factors, for which we have no control or awareness, can affect the pacemaker circuit. One of these is synaptic input from other brain regions. The most general input, a random (Poisson) activation of synapses, is experimentally observed in virtually every in vivo recording as noise. Let us consider what happens when a low (subthreshold) level of background synaptic noise arrives on the pacemaker circuit. In Fig. 2B we superimpose typical somatic membrane potential from 3 simulations. Under control conditions (Fig. 2B, control trace), its activation elicited an AP with a \( \approx 5 \) ms latency. A purely excitatory background noise of \( 0.15 \pm 0.04 \) nS (Fig. 2B, exc. trace) reduced the latency to \( \approx 3 \) ms. When an inhibitory noise of \( 0.25 \pm 0.1 \) nS was also added (Fig. 2B, exc. + inhib. trace) the latency increased up to \( \approx 8 \) ms. These latency shifts will affect each AP. For stronger noise levels, as shown in Fig. 2C, with respect to the control simulation, an excitatory background (Fig. 2C, exc. trace, 1.5 \pm 0.5 nS) elicited additional spikes. When an inhibitory noise (4 \pm 1.2 nS) was added (Fig. 2C, exc. + inhib. trace) spikes were missed. Thus, a background synaptic noise can alter the overall pacemaker frequency. We went one step further, testing if a correlation could be established between the quality and amount of the noise and the complexity of the cognitive load. In fact, a cognitive load requires to focus the attention on a non-temporal task, and there are a number of studies suggesting that excitation and inhibition are the driving forces of selective attention (Ghatan et al., 1998). We speculated that the process to focus the attention on a given task would generate a mostly inhibitory background activity on all those brain regions that are not involved with that task, including the pacemaker circuit for a non-temporal task. In fact, when we reproduced the experimental paradigms of Fig. 1, we found that the background inhibitory noise...
was able to reproduce the qualitative effects of an increasing cognitive load on production of different lengths intervals (Marmaras et al., 1995), as shown in Fig. 3A. It should be noted that, in these simulations, the excitatory noise was fixed (1 ± 0.3 nS). The level of the inhibitory noise was the only parameter needed to take into account this kind of experiments. As shown in Fig. 3B, the model was also able to reproduce the experimental finding of a negative relationship between a cognitive load and the estimation of a time interval (Zakay 1993, Fig. 1B).

The reason why a background noise can give a reasonable representation of the effects of attention (and thus of cognitive loads) on the timekeeping systems can be better appreciated in the schematic representation of Fig. 4. In this simulation, because of the inhibitory noise, some synaptic signals generated by the feedback circuit were not transformed in actual pacemaker APs. Let us consider the task to estimate an 8 t.u. interval, assuming that the pacemaker generates 1 AP/t.u. In an interval estimation paradigm, the experimenter gives a start and a stop after 8 t.u. (Fig. 4, start_e and stop_e marks) and the estimation is carried out counting the spikes within the interval, giving (in this case) an estimate of 6 t.u., lower than the real value of 8 t.u. In producing the same 8 t.u. interval, a subject would start by himself and wait to stop until eight spikes are generated (Fig. 4, start_p and stop_p marks). However, the external clock would measure a longer interval (≈14 t.u. in this case), as observed in the experiments, in agreement with the predictions of the psychological attentional model, and consistent with our common everyday experience. It is easy to see (not shown) that an excitatory noise would generate additional spikes, improving both production and estimation of an interval, and could represent the effects of attention devoted to temporal-related processing activities, such as time counting strategies.

4. Discussion

How the brain keeps track of the elapsed time on scales from seconds to hours, is a debated issue. This is mostly due to the lack of direct experimental evidence in favor of one particular model. Here we would like to point out that, in any case, one or more neuronal circuits need to be

Fig. 3. (A) Model’s production of time intervals of different lengths as a function of the background inhibitory synaptic noise. The excitatory noise level was 1 ± 0.3 nS in all cases. (B) Model’s production and estimation of a 12 s interval as a function of background noise. Errors were smaller than symbols size and are not shown.
used as timekeepers. They could be implemented as simple pacemakers (Treisman, 1963), distributed interval timing systems (Ivry, 1996; Bugmann, 1998), or a more general representation of temporal events based on past experiences (Grossberg and Merrill, 1996), but common sense and experimental findings suggest that the evaluation of time intervals always require attentional resources (Zakay and Block, 1996).

Attention-based facilitation of task-relevant processes, and inhibition of unattended activities modulating competitive interactions among neurons, have been found using functional imaging techniques (Ghatan et al., 1998; Kastner et al., 1998) and intracellular recordings in vivo (Reynolds et al., 1999). Using a common mechanism for perception and production of temporal intervals (Ivry and Hazeltine, 1995), we have shown that the effects of the attentional network activity (activated by the cognitive loads) could be modeled by just increasing the inhibitory background synaptic noise. Thus, the qualitative results of our model are not affected by the particular values for the pacemaker parameters (such as, conduction delay, oscillation frequency, synaptic localization, passive or active membrane properties, etc.) that we have used in this paper. Within this framework, the experimental findings on intervals evaluation could be interpreted in terms of the amount of attention devoted to the elapsing time. The excitatory and inhibitory components of the noise would be the consequences of the activity generated by the attentional network to carry out concurrent competitive tasks, eventually correlated with the conscious selection of the stimuli to attend or tasks to execute (Posner, 1994; Posner and Rothbart, 1998). Thus, when involved in interesting activities we focus our attention on them and forget about time. The attentional system inhibits all unattended activities, the internal timer slows and the estimation of elapsed time is lower than the real value. Puzzling experimental data showing that both higher and lower than normal body temperature result in shorter interval productions (Hancock, 1993), could be interpreted as attentional resources directed more to the elapsing time rather than to the obnoxious sensorial stimuli.

Modeling of other experimental observations, such as the large variability and its dependence on the interval duration, could give useful insights on how humans process temporal information. Mechanisms such as internal representations of the target intervals (Ivry, 1996) or memory effects (Gibbon et al., 1997) have been proposed to explain these findings. However, the experimental values for the coefficient of variation in timing tasks for humans and animals across a number of tasks (Gibbon et al., 1997) do not show a clear trend. For this reason, we have chosen not to take into account these effects in the present model.
Finally, although in this work we have used a pacemaker, the results of our model could have a general validity, suggesting how the attentional networks could be involved at the single neuron level. Under a non-temporal cognitive load, any timekeeper system would be affected by the inhibitory activity directed toward unattended processes by the attentional network, independently of number, localization or implementation.

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